Project Abstract

On Long-term Consequences of Selfish Behavior: A Game Theoretic Approach to Host-Pathogen Coevolution

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Overall Mission/Objective

Optimal management strategies constitute underlying themes of conservation ecology and economics, and exploring the junction between these two disciplines is both important and of broad interest. Humans, as well as other social species, face trade-offs between self and group "interest", with efficient cooperation often critical for long term survival. An interesting case of this problem is that of human-disease interactions, since not only must we, as humans, manage our natural enemies, but these enemies must also manage us.

Such coevolutionary dynamics can be described by a multi-layered game, with strategic interactions between the individual hosts, as well as between the hosts and their pathogens. Despite the advantages the hosts derive from contact with one another, there are also costs in the form of exposure to infectious diseases, and so, the (Nash) noncooperative equilibrium level of contact between selfish host individuals will necessarily be greater than the level that would maximize the group fitness/welfare. What is more, these higher levels of contact are hypothesized to influence the evolution of the pathogen, bringing us to the other dimension of this game: optimal virulence. Fitness-maximizing levels of virulence depend on the levels of contact between the hosts, which in turn, are partly determined by the virulence of the pathogen.

Such dynamics may result in significantly higher levels of infection, and higher rates of mortality for infected individuals, than would be selected for if the hosts were to cooperate. The method by which modern rational humans may enforce cooperation is through public health policies that facilitate avoiding infection, and the ultimate objective of our analysis is to suggest improved policies that account for the long-run consequences of these health authorities' actions.

We have thus proposed to develop models of host-pathogen coevolution that combine variants of the classical S-I dynamic model with the structure of game theory. This may allow us to predict the welfare effects of various public policies.

Our research agenda has two main stages. The first stage lays the theoretical groundwork for the coevolution of host social behavior and pathogen virulence. This stage has been completed and the results are currently in press in the science journal *Evolution*. Our

second stage will build from some of the theoretical work in the first stage, but will focus specifically on a human-malaria game.

Progress and (Preliminary Outcomes)

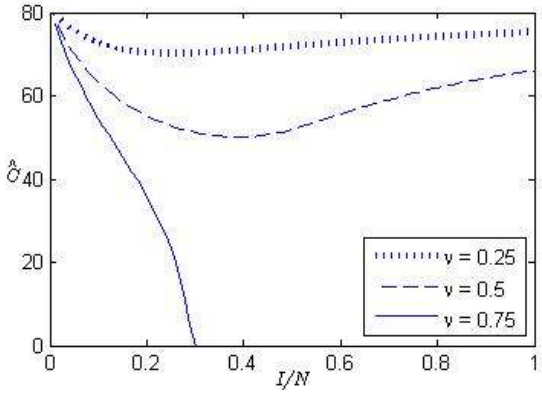
Higher disease prevalence can induce greater sociality; a game theoretic coevolutionary model

(Bonds, M.H., D.C. Keenan, A.J. Leidner, and P. Rohani, 2005) *Evolution*, in press.

There is growing evidence that communicable diseases constitute a strong selective force on the evolution of social systems. Indeed, it has been suggested that infectious diseases may determine upper limits of host sociality by, for example, inducing territoriality or early juvenile dispersal. Here, we used game theory to model the evolution of host sociality in the context of communicable diseases. Our model was then augmented with the evolution of virulence to determine coevolutionarily stable strategies of host sociality and pathogen virulence.

We show that, as generally hypothesized, exogenously increasing disease prevalence from initial low values results in a decrease in the evolutionarily stable rate of contact. However, for a large range of the parameter space, the optimal contact rate increases as prevalence rises past a threshold level. In other words, though the disease never causes sociality to rise above the disease-free optimum, higher disease prevalence can actually *induce* greater sociality. This result is similar to that of van Baalen (1997) who finds that the optimal investment in immunity rises and then falls as infection probabilities rise. The reason for both of these results is that the costs of the host's response to the disease, in terms of higher mortality, eventually overwhelm the benefits of those responses when infection is sufficiently difficult to evade. In our model, this means that the host would then evolve greater sociality at high levels of disease prevalence. An exception to this relationship is when the pathogen is sufficiently virulent to eliminate the value of contact after infection (Figure 1). In this case, the optimal contact will fall monotonically as prevalence rises.

Figure 1

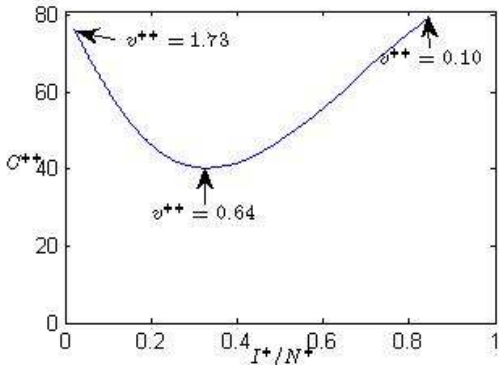


The optimal contact rate is presented over a range of exogenously determined rates of disease prevalence, with different values of pathogen virulence. At relatively low levels of

virulence, the ES contact rate responds nonmonotonically to disease prevalence. At high levels of virulence, the ES contact rate responds negatively to disease prevalence

Finally we considered pathogen coevolution. We show that lower contact increases pathogen virulence, counter to Ewald's (1994) hypothesis. This is because, as contact rates fall from the disease-free optimum, so does host survival, lowering the benefits for the pathogen of preserving the host, and increasing the advantages of being transmissive. As we alter various parameter values, we continue to find a U-shaped relationship between the CoES level of host sociality and the equilibrium disease prevalence (Figure 2). We also find a U-shaped relationship between the CoES level of host sociality and pathogen virulence.

Figure 2



The coevolutionary relationship between contact and disease prevalence is U-shaped across background mortality rates.

Evolution of a disease-driven poverty trap

One billion people in the world currently live on less than $1 per day, which is considered "extreme poverty" and has special economic consequence. $1 per day places people on the edge of the ability to sustain their lives. And living on such an edge precludes saving for the future. In the absence of saving, resources (specifically "capital", in economic jargon) cannot accumulate, greatly confounding the prospects of raising standards of living. As a result, one sixth of all humans are roughly as poor today as their ancestors were thousands of years ago. How is that possible? A plausible explanation is that the world's poorest individuals are stuck in a disease-driven "poverty-trap" (Sachs, 2005). The basics of this idea are simple. Both the economy and public health serve as essential positive determinants of each other: economic activity requires healthy labor, and disease prevention and treatment require economic resources. So when an infectious disease spreads throughout a population, the effective labor force shrinks, which further debilitates the ability of the population to afford measures to fight off diseases. What makes things even more complex is that both income and health are determined not just by the behavior of the individuals, but also by their geographic and

social context (remember, it is *infectious* diseases – those that are transmitted by neighbors – that are the chief villains). Thus, sustaining income growth for one individual requires sustained health of his/her community. The most glaring modern example of this is AIDS in Africa where rural communities are commonly known to have lost the entire core of their local economies – working-age people. But historically, the biggest culprit of the poverty trap may well be Malaria. (Sachs, 2002).

To date, the disease-driven poverty-trap has been treated mostly as an economic question (Sachs, 2002). But diseases are not static forces that can be thrown linearly into economic growth models. The transmission of malaria, for example, is determined by the population dynamics of an animal – the *Anopheles* mosquito – which is itself involved in an evolutionary relationship with the disease that it carries. What makes this topic especially interesting is that human behavior has predictable consequences for the evolution of both the mosquito and the pathogen. Furthermore, the relationship between this behavior and economic growth is predictable. Due to basic interventions such as glass windows and bednets (to name a few), wealthy people tend to be less exposed to the transmission of vector-borne diseases. This lower exposure is analogous to a decrease in host "contact" and we can therefore rely on the theoretical framework developed in the first stage of our project. Further development of this economic-growth malaria "game" constitutes our research for the remaining duration of the grant.

Broader Impacts; teaching, training, and learning

Our research budget has been almost entirely allocated towards the development and training of our graduate research assistant, Matthew Bonds, and to a lesser degree, an undergraduate research assistant, Andrew Leidner. Under our close supervision, Matt has been delegated the primary responsibilities of developing the project. Matt has presented our project at the 2005 *Ecological Society of America* conference in Montreal, Quebec, as well as at the 2005 *Southeastern Ecology and Evolution Conference*, in Athens, Georgia, where he received an award for the best oral presentation. He also assists in managing our undergraduate assistant Andrew Leidner, who works with us for 15 hours per week. Since joining our group, Andrew has learned to program simulations of disease evolution in C++ and is a coauthor of our article in *Evolution*.

Finally, we are currently arranging for Matt to visit Andrew Read at the University of Edinburgh in Scotland where he can learn directly from Read's malaria experiments. While in the United Kingdom, Matt also plans to visit other prominent scientists who work on pathogen evolution. These scientists include Steve Sait of the University of Leeds, Mike Boots of the University of Sheffield, and Julia Gog of the University of Cambridge.

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